Diagnosis and Treatment of Clinical Rumen Acidosis

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KEYWORDS

• Acidosis • Rumenitis • Liver abscessation • Laminitis

KEY POINTS

- Although classically considered a disease of cattle fed in confinement, rumen acidosis is a common cause of morbidity and mortality in both small and large ruminant populations.
- Feeding and management practices that lead to consumption of large amounts of feed containing readily fermentable carbohydrates precipitate clinical disease.
- Sequelae to rumen acidosis include laminitis, rumen ulceration, liver abscessation, and thromboembolic respiratory disease, each of which can have a greater impact on animal health and well-being than the primary disease process.
- Treatment of the individual animal with rumen acidosis focuses on correction of volume deficits, supplementation of alkalinizing agents, restoration of a normal rumen microenvironment, and management of secondary complications.
- Prevention of rumen acidosis is centered on restricting access to feeds containing readily fermentable carbohydrates to which animals are not accustomed, gradually introducing feed containing concentrates over a period of weeks, and addressing management practices that promote aggressive eating behavior.

INTRODUCTION

Clinical rumen acidosis remains a major cause of morbidity and mortality in modern ruminant production systems. Survey data from feedlot cattle have revealed that approximately 4.4% of cattle placed on feed are diagnosed with digestive problems.[1] Similarly, it is estimated that anywhere from 14% to 42% of deaths in feedlot cattle are due to digestive disorders, making them the second leading cause of mortality in feedlots.[2,3] Acute rumen acidosis represents an economically important loss to the beef industry. It has been estimated that the average cost of treating cattle with acidosis

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averages $10/head and, with an estimated 10.7 million cattle on feed as of December 1, 2016, this would translate to about $4.6 million in treatment costs alone.[4]

Although commonly viewed as a disease of cattle on high-concentrate rations, rumen acidosis has also been reported in sheep, goats, and New World camelids. Although few data are available evaluating the true incidence and impact of acidosis in these populations, it is thought that the disease occurs less frequently because of a combination of factors that include differences in behavior, feeding practices, and forestomach physiology. Nevertheless, clinical rumen acidosis can be responsible for considerable morbidity and mortality in some small ruminant populations, making recognition of the disease important for any practitioner.

This text reviews available data regarding the pathophysiology, clinical signs, diagnosis, treatment, and prevention of clinical rumen acidosis in cattle, sheep, goats, and New World camelids.

PATHOPHYSIOLOGY

Often viewed as the most dramatic form of the forestomach fermentative disorders, clinical ruminal acidosis occurs when excessive levels of organic acids accumulate in the rumen, resulting in a rumen fluid pH of less than 5.2.[2,5] A common scenario for the development of clinical rumen acidosis is the excessive consumption of rapidly fermentable carbohydrates by ruminants that are unadapted to a high-concentrate diet. As a result, clinical rumen acidosis is often seen in the early feeding period when newly received feedlot cattle, accustomed to a primarily forage-based diet, are introduced to a primarily concentrate-based ration and stepped up too rapidly.[2,3] Similar signs can also develop when concentrate-adapted ruminants are fed more concentrate than their ruminal microbial population can handle. This situation might occur following a feeding error, overprocessing of grain, changes in ration moisture, or when there is excessive competition for feed within an animal population.[5,6] Excessive feeding of rapidly fermentable carbohydrates, commonly referred to as "grain overload," is the classic scenario leading to clinical rumen acidosis. It is important to remember, however, that excess grain consumption is not essential to the development of the syndrome, because excess consumption of any rapidly fermentable carbohydrate (apples and other fruits, bakery waste products, incompletely fermented brewery products, and standing green corn) is capable of providing the necessary substrate for the development of clinical disease.[6] In fact, the authors have seen clinical rumen acidosis in mature does following consumption of excessive amounts of animal crackers given by the owners as treats.

Regardless of the initial inciting cause, the pathogenesis of clinical rumen acidosis is the same. Ruminal bacteria that digest starches and sugars proliferate and increase their rate of carbohydrate fermentation. In the normal animal, or in animals with mild clinical disease, rumen buffering capacity and volatile fatty acid (VFA) absorption match the rate of carbohydrate fermentation. In this scenario, the pH within the rumen will stay in a normal range between 5.6 and 6.9, with the higher pH range being more common in New World camelids.[5,7–9] However, when production of VFAs and lactate exceeds the rate of absorption, rumen pH will begin to drop. VFAs and lactate increase in concentration within the rumen fluid and are subsequently absorbed into the systemic circulation.[10] Although numerous microorganisms have been implicated in the development of disease, the primary bacterium thought to be associated with the progression of clinical signs is *Streptococcus bovis*. *S bovis*, because of its rapid rate of division, ability to produce more ATP per unit time, and tolerance of a pH <5.5, is the microorganism that sets the stage for acid production and worsening of symptoms.[7]

Nevertheless, *S bovis* is intolerant of a pH >4.5. As pH decreases, lactate production by *S bovis* decreases, and the growth of *S bovis* is slowed.[7] At this point, the *Lactobacilli* become the dominant microbes present in the rumen and further serve to depress ruminal pH.[2]

There are 2 chiral forms of lactate produced in the rumen: D and L. The L isomer is produced by both mammalian and microbial cells, whereas the D form is produced primarily by microbes.[5] The L-lactate is easily metabolized by mammalian cardiac and hepatic tissues. D-Lactate is not metabolized nearly as efficiently as L-lactate and accumulates in circulation.[5] For this reason, a more appropriate term for clinical rumen acidosis would be acute D-lactic acidosis.[11] Nevertheless, regardless of the form, lactate appears to be less readily absorbed across the rumen wall than are VFAs. Indeed, VFAs tend to be much weaker acids than either D- or L-lactate and serve as buffers in the rumen fluid. This phenomenon contributes to many of the VFAs existing in the nondissociated state, a factor that enhances their absorption into the systemic circulation.[12,13] Despite this, enough lactate is absorbed in the forestomach and more distal portions of the digestive tract to cause acidemia. Furthermore, many VFAs are transformed into lactate by the rumen wall to further add to the acid load in the bloodstream.[8,12] In addition, as rumen pH further declines, lactate starts to become the dominant organic acid present in significant amounts in the rumen. It is thought that the decrease in numbers of VFA-producing microbes and increased activity of pyruvate dehydrogenase in the acidic rumen environment promote the further accumulation of lactate.[5,7]

D- and L-Lactate are powerful corrosive agents that can cause severe damage to the rumen epithelium. In addition, lactate and VFAs are osmotically active. Increased rumen osmolarity decreases absorption of lactate and VFAs, creating a cycle that perpetuates buildup of these compounds and a continued drop in pH.[5,13] With the continued accumulation of these compounds and further increases in rumen fluid osmolarity, the rumen epithelium is further disrupted. Yeast and fungi that are resistant to highly acidic environments readily colonize the denuded sites and contribute to the development of mycotic rumenitis and omasitis. In addition, organisms such as *Fusobacterium necrophorum* are able to invade the bloodstream and spread to the liver. In fact, rumen acidosis is thought to be one of the inciting causes for the development of liver abscesses in ruminants. In addition to their effects on the rumen, the osmotic pressure of these agents causes systemic dehydration and hypovolemia by pulling fluid from the circulation into the rumen, resulting in a reduction in tissue perfusion.[5,14] The loss of circulating blood volume leads to cardiovascular collapse, reduced renal perfusion, and anuria. Reduced peripheral circulation also leads to anaerobic cellular metabolism and systemic acidosis.

In addition to lactate, there are many other compounds produced by rumen microbes that can be deleterious to multiple organ systems. Some of these deleterious compounds include endotoxins and histamine.[2] Even in the normal state, endotoxin can be found in the rumen contents, and in these situations, it exists without negative systemic effects on the animal.[7] However, endotoxin concentrations will increase in the rumen of animals on a concentrate-based diet.[15] If these animals become acutely acidotic, the acidic environment within the rumen fluid can cause microbial death and release of endotoxin in large quantities all at once.[7] The ability of the normal, intact rumen to absorb endotoxin into the systemic circulation has been questioned, but some researchers have documented endotoxin present in the bloodstream following an acidotic event.[7,16–18]

In addition to endotoxin, histamine is also known to accumulate in the acidotic rumen. Histamine-producing microbes do not exist in large numbers in animals being fed a

forage-based ration.[7] However, they proliferate rapidly in cattle on concentrate-based rations, and even more so in an acutely acidic environment. *Allisonellla histaminiformans* thrives at low pH, produces large quantities of histamine, and is thought to be the major player in ruminal histamine production.[19] Histamine is not well absorbed by the healthy rumen wall; however, when the ruminal epithelium is damaged as occurs in acidosis, histamine can be absorbed through the rumen wall and into the systemic circulation.[20] Histamine can also be absorbed via the small intestines.[6] There are many effects that histamine is thought to have systemically that may further intensify the symptoms of acute acidosis, including vasodilation and arterioconstriction, and increase vascular permeability.[20,21]

These effects are likely partly responsible for one of the most common sequelae of acidosis in ruminants: laminitis. It is thought that this combination of effects causes blood pressure to increase in capillaries and edema, resulting in swelling, hemorrhage, and even rupture of the vessels.[21] The interruption of blood flow to the tissues in the hoof can thus result in local ischemia and damage to the corium.[21] Laminitis is commonly seen with acidosis in cattle and sheep, but less so in goats.[22] In acidosis of mild severity, animals can experience a transient lameness that seems to resolve following correction of the acidotic event. However, animals experiencing a severe acute case can have more serious lesions, and animals experiencing subacute acidosis can develop subclinical or chronic lesions because of long-term damage to the tissues of the hoof.[21]

CLINICAL SIGNS

The signs of acute rumen acidosis vary according to the type and amount of feed ingested, amount of time since feed ingestion, and severity of physiologic derangements. It is often helpful to classify the manifestations of acidosis into subacute, acute, and peracute forms.[23] In subacute cases, affected animals remain bright, alert, and responsive but may have transient anorexia with signs of mild to moderate dehydration. In these cases, rumen motility is reduced, but diarrhea and signs of abdominal pain are inconsistent.[6,22] In lactating animals, milk production will often be decreased. In some cases, abortions, stillbirths, and premature births may be the only signs seen by caretakers, and the authors have been involved in multiple investigations where abortion storms were the only signs of acidosis noted following the inclusion of bakery waste in the ration of pastured cattle.

In acute cases, affected animals can be found severely obtunded and ataxic. The rumen will usually appear distended, and sloshing of fluid within the rumen is heard on auscultation with abdominal ballottement. Ruminal contractions will be weak to absent.[22] Anorexia will be present, along with profuse, watery, foul-smelling diarrhea. Feces will often be gray in color and may contain bits of undigested grain. In some cases, frank blood may be noted in the feces. In the early stages of disease, rectal temperatures are consistently increased. However, with clinical progression, hypothermia is often detected.[24,25] Tachycardia and tachypnea are present in many cases, with respirations often characterized as shallow.[23] Dehydration and/or hypovolemia are present and are manifested as sinking of the eyes into the orbits, prolonged skin tenting and capillary refill times, delayed jugular filling, weak peripheral pulses, and cold extremities. Neurologic manifestations of disease are seen in many cases and include obtundation, blindness, head pressing, opisthotonus, and altered gait. One particularly useful test is evaluation of the palpebral reflex. There is a very good correlation between the extent of palpebral reflex depression and severity of D-lactic acidosis, and this test might be helpful in categorizing disease severity and monitoring response to therapy.[26,27]

In the peracute form of the disease, animals might be found dead with few or no premonitory signs. In some instances, animals are found recumbent and comatose with the head tucked into the flank. In these severe cases, prognosis is generally poor, and death occurs within a matter of hours.

CLINICAL PATHOLOGY

Multiple diagnostic tests are available to the practitioner evaluating an animal with rumen acidosis. Rumen fluid analysis, complete blood count (CBC), serum or plasma biochemical profiles, blood-gas analysis, and urinalysis have been used to confirm a diagnosis, evaluate disease severity, and assess physiologic derangements. Although not often necessary to confirm a clinical diagnosis, these ancillary diagnostic tests might be of use in determining a prognosis for the individual animal and the degree to which the patient might need to be supported with therapeutic interventions.

Analysis of rumen fluid is one of the most useful diagnostic tests available and should be performed on any animal where rumen acidosis is suspected. Rumen fluid can be collected via passage of an ororumen tube or ruminal paracentesis. As a general rule of thumb, the fluid should be evaluated promptly after collection for color, consistency, odor, pH, and microbial activity (Table 1). Normal rumen fluid should be olive or brownish-green and slightly viscous with an aromatic odor. In animals with acidosis, rumen fluid might be milk gray with a putrid odor and watery consistency. In animals on a roughage diet, rumen pH should be 6 to 7, whereas those on high-grain diets might be 5.5 to 6. Regardless, a rumen fluid pH of less than 5.5 is consistent with a diagnosis of rumen acidosis. Microscopically, rumen fluid from animals with acidosis will have decreased numbers and activity of protozoa. Usually, absolute numbers of large and medium protozoa species are decreased. Gram stain can be used to evaluate bacterial diversity and might reveal a shift from gram-negative organisms to a population of predominantly gram-positive organisms.

Hematologic findings in ruminants with acidosis are rarely specific to the condition and generally reflect underlying inflammatory processes. A CBC will often reveal evidence of dehydration and systemic inflammation. Often, animals with acute rumen acidosis will have polycythemia and a neutropenia with a left shift. In animals with longer-standing disturbances, a neutrophilic leukocytosis with hyperfibrinogenemia might be seen.

A serum or plasma biochemical profile might be useful in evaluating organ function, evaluating electrolyte and acid-base homeostasis, and establishing a prognosis. Changes in the biochemical profile are dependent on disease duration and severity

Table 1
Characteristics of rumen fluid in clinical normal ruminants and ruminants with acute rumen acidosis

Parameter	Normal	Rumen Acidosis
Color	Olive to brownish-green	Yellow to gray
Consistency	Viscous	Thin and watery
Odor	Aromatic	Fetid
pH	5.5–7 depending on diet	<5.2
Protozoal activity	Large, medium, and small forms, actively moving	Reduced numbers, little active movement
Gram stain	Predominantly Gram (−)	Predominantly Gram (+)

and include azotemia and hyperphosphatemia, elevations in hepatic enzymes, hyperkalemia, hyperchloremia, mild hypocalcemia, and metabolic acidosis. Azotemia and hyperphosphatemia result from renal compromise associated with hypovolemia and systemic inflammation. Elevations in hepatic enzymes might be reflective of hepatic damage associated with decreased perfusion or bacterial colonization of hepatic tissues. Hyperkalemia is associated with decreased distal tubular flow and accumulation of potassium in circulation.[28] Hyperchloremia and metabolic acidosis are reflective of the accumulation of acid in circulation and titration of the body's buffer systems.

Blood-gas analysis can be helpful in the development of a treatment plan and assessing the severity of acidosis. Ruminants with acidosis will have decreases in plasma pH, elevations in anion gap, and decreases in base excess. In many cases, plasma lactate concentrations are elevated. It is important to note that there might be a discrepancy between plasma lactate concentrations and degree of change in the anion gap and base excess on most commercial blood-gas analyzers. This discrepancy likely reflects the fact that animals with rumen acidosis experience accumulations of both D- and L-lactate in circulation, and most analyzers only detect L-lactate. It is important to remember that D-lactate is an important contributor to the clinical presentation of ruminants with acidosis, and, even though it is not routinely measured, the clinician should not overlook the importance of D-lactate to the pathophysiology of rumen acidosis when designing treatment protocols.

DIAGNOSIS AND PROGNOSIS

Diagnosis of clinical rumen acidosis is based on history of exposure to offending feedstuffs, clinical signs, and ancillary diagnostic tests, particularly rumen fluid analysis. Although the clinical presentation of rumen acidosis can mimic the presentation of other common diseases (mastitis, metritis, peritonitis), the animals' signalment, the lack of signs referable to other body systems, and results of rumen fluid analysis usually can assist the clinician in making an accurate diagnosis and developing an appropriate therapeutic plan. Therefore, a thorough examination of every body system is necessary to rule out other disease syndromes and is an essential component of managing animals with acidosis.

The prognosis for animals with rumen acidosis depends on the duration and severity of clinical signs. Ancillary diagnostic tests play an important role in the assessment of disease severity and development of treatment plans, and their use should be considered if economically feasible. Animals with a blood pH of less than 7.2, rumen pH less than 4.5, severe central nervous system signs, and anuria are less likely to survive than animals with less severe changes. Similarly, animals with peracute disease onset are unlikely to respond to treatment.[23]

TREATMENT

Animals with mild clinical disease might recover with little to no specific care. In animals with more severe clinical disease, specific therapy is necessary and is focused on correction of plasma volume deficits, assessment and treatment of local (rumen) and systemic acid-base disturbances, restoration of a normal rumen microenvironment, and treatment/prevention of potential secondary complications.

Volume deficits are assessed during the initial physical examination, and their severity can be determined in several ways. Although not experimentally validated in mature cattle, the use of eyeball recession has been shown to correlate well with degree of dehydration in calves.[29] The formula for determination of % dehydration based on eyeball recession is as follows:

Degree of recession in millimeters (mm) × 2 = % dehydration

Thus, an animal with the eyes recessed 5 mm in the orbits would be approximately 10% dehydrated. Other methods of determining fluid deficits include the use of skin tenting times, capillary refill time, jugular filling time, and clinicopathologic variables (Table 2). It should be remembered, however, that many of these parameters can be misleading if not interpreted in light of physical examination findings.

Once % dehydration is assessed, the patient's fluid deficits must be estimated and can be done using the following formula:

% dehydration × body weight (BW) in kg = Fluid deficit in liters (L)

As an example, a 500-kg steer that is 10% dehydrated would require 50 L of fluid to replace existing fluid deficits.

Another consideration when developing a fluid therapy plan for a ruminant with grain overload is assessment of the severity of acid-base disturbances by determining plasma HCO_3^- deficit. The amount of HCO_3^- required to fully replace the total body HCO_3^- deficit can be calculated using the following formula:

0.3 × BW (kg) × HCO_3^- deficit = Amount of HCO_3^- required in milliequivalents (mEq)

Again, a 500-kg steer with a plasma HCO_3^- concentration of 5 mEq (HCO_3^- deficit = 25 − 5 = 20) would require 3000 mEq of $NaHCO_3$ to correct the entirety of the calculated deficit.

Dehydration and acidosis are both important considerations for the practitioner when choosing a fluid type for use in a patient with clinical acidosis. Both contribute to morbidity and mortality, and neither factor should be overlooked when choosing a resuscitation fluid. In animals with severe clinical disease, balanced electrolyte solutions should be used to replace existing fluid deficits and provide for maintenance fluid needs. It should be noted that these fluids are usually only being used to address plasma volume deficits and not systemic acid-base disturbances. It is the authors' preference to replace fluid deficits over a period of 24 hours in animals with pure dehydration (sunken eyes and prolonged skin tent with adequate jugular filling and capillary refill time [CRT]). In animals with hypovolemia (prolonged jugular filling, cool extremities, prolonged CRT), the authors use the fluid challenge model whereby 20 mL/kg boluses are given over a 20- to 30-minute period.[30] The patient is reassessed following each bolus and can receive up to 4 boluses before significant fluid overload is a concern. Another option for resuscitation of large ruminants with significant volume deficits is hypertonic saline (HS). HS (7.2%) can be administered at a dose of 4 mL/kg over a period of approximately 10 minutes.[31] It is thought that each milliliter of

Table 2
Indicators of dehydration and hypovolemia in ruminants with acute rumen acidosis

Parameter	Normal Value
Degree of enophthalmos	<2 mm
Skin tenting time	<1 s
Jugular filling time	Rapid, no delay after compression of vein
Capillary refill time	<2 s
Temperature of extremities (distal limbs, ears) and strength of pulses	Warm to touch, strong pulses

HS will expand plasma volume by 3 to 4 times the amount infused.[32] It is important to remember that the effects of HS are transient and that these solutions must be followed by additional oral or intravenous fluids at a rate of 5 to 10 L per liter of HS infused.[32]

Hypertonic and isotonic solutions of $NaHCO_3$ are commercially available and can be used in conjunction with other fluids to address systemic acidosis. As a general rule of thumb, roughly half the calculated $NaHCO_3$ deficit should be replaced initially, with the remaining half being replaced over the course of 24 to 48 hours. Although the above method may be useful in hospital settings, the diagnostic tests needed to calculate $NaHCO_3$ deficits might not be available in the field. Therefore, protocols have been developed that rely more on a standardized set of recommendations than laboratory diagnostics. One such protocol suggests the use of a 5% solution of $NaHCO_3$, given at a rate of 5 L over 30 minutes for a 450-kg animal, followed by 1.3% $NaHCO_3$ administered at a rate of 150 mL/kg of body weight over 6 to 12 hours.[6] In recent years, many investigators have begun to evaluate 8.4% $NaHCO_3$ solutions for their utility in treating neonatal ruminants with moderate to severe D-lactic acidosis. In these settings, these solutions are safe and effective in treating acidosis and increasing plasma volume.[33,34] Although not specifically evaluated in large ruminants with acute rumen acidosis, 8.4% $NaHCO_3$ might be useful, particularly in field settings where time and resources are limited. $NaHCO_3$ (8.4%) should be administered at a rate of 5 mL/kg of body weight over a period of 10 to 20 minutes.[33,34] Like HS solutions, 8.4% $NaHCO_3$ should be followed with isotonic intravenous or oral fluids to further expand plasma volume and prolong the duration of effect.[33,34]

Restoration of the rumen microenvironment involves removal of acidic rumen contents, administration of rumen buffers, and transfaunation. Removal of abnormal rumen contents can be accomplished in 1 of 2 ways: rumenotomy or rumen lavage. The decision to perform a rumenotomy to remove acidic rumen contents should be made based on the severity of the case, the chances for recovery, and the economic value of the animal. In very severely affected animals of high economic value, rumenotomy can be a highly effective treatment. The surgical techniques to perform the procedure are discussed in detail elsewhere.[25,35] In less severe cases or in animals with lower economic value, removal of the acidic rumen contents can be accomplished by repeated flushing of the rumen with warm water through a large-bore stomach tube. In cattle that are depressed but still standing, this technique allows for a nonsurgical method of removal of rumen contents. This therapy is not well suited to camelids and small ruminants because the diameter of the ororumen tube required precludes passage down the esophagus. With this technique, a large volume of water is pumped into the rumen, and the rumen contents are allowed to flow out by means of pressure and gravity. It is important that an ororumen tube with sufficient diameter be used so that it does not easily become clogged with ingested feedstuffs. Thus, a tube with internal diameter of 1 inch is usually required.[6] Any animals receiving either a rumenotomy or rumen lavage should be transfaunated with rumen fluid taken from a donor on a normal diet. Although the ideal amount of rumen fluid is not known, other investigators have suggested the administration of anywhere from 3 to 10 L in large ruminants.[36] In small ruminants, 2 to 4 L of rumen fluid is usually sufficient.[25]

In animals not undergoing rumenotomy or lavage, oral administration of a buffer is an indispensable and helpful step in correcting ruminal acidosis. A magnesium hydroxide solution is preferable to $NaHCO_3$, because placing $NaHCO_3$ into a highly acidic environment will result in the release of CO_2 and could potentially result in bloat. Magnesium hydroxide should be given at a dose of 1 g/kg of body weight and dissolved in enough water to ensure its dispersal throughout the rumen.[6] It is important

to note that the administration of magnesium hydroxide to normal animals is not without consequences.[37] In these patients, magnesium hydroxide will disrupt the rumen microflora, contribute to systemic alkalosis, and cause hypermagnesemia.[37] Thus, rumen acidosis should be confirmed before these agents are administered to any patient.

Numerous sequelae have been associated with clinical rumen acidosis and include polioencephalomalacia (PEM), rumenitis, liver abscessation, laminitis, and vena caval thrombosis. Ancillary therapies are focused on the prevention of longer-term effects of acute ruminal acidosis. One such therapy is the administration of thiamine for the prevention of PEM. The syndrome has been observed in all major domestic ruminant species as well as New World camelids.[9] It is not unusual to see PEM following an acute acidotic event, although the exact mechanism is not fully understood and is discussed in more depth elsewhere.[38] It is thought that PEM following acidosis could be due to the reduction in numbers of thiamine-producing bacteria in the acidic rumen or an increase in the activity of ruminal thiaminases.[38–40] The suggested treatment of PEM in cattle is 10 mg/kg of thiamine given intramuscularly (IM) or subcutaneously, every 8 hours for 3 days in symptomatic animals.[41] The use of dexamethasone, nonsteroidal anti-inflammatory drugs, dimethyl sulfoxide, or diuretics for the treatment and/or prevention of PEM is not supported by the available literature.[40] Another ancillary therapy includes administration of oral or parenteral antimicrobials to prevent liver abscesses and vena caval thrombosis. Unfortunately, the use of antimicrobials in animals with acute acidosis is controversial. Although some have suggested oral administration of penicillin in animals with acidosis, it is not currently recommended because it may have deleterious effects on the rumen microbes and could interfere with reestablishment of a normal bacterial population.[25,35] Nevertheless, parenteral antimicrobials might have some benefit in reducing the development of liver abscesses, and antimicrobials with a spectrum of activity against *F necrophorum* could positively impact patient outcome. The authors routinely recommend the administration of procaine penicillin G at a dose of 22,000 IU/kg IM or ampicillin trihydrate at a dose of 11 mg/kg IM once daily for 3 to 5 days to animals with acute rumen acidosis.

PREVENTION

Prevention of rumen acidosis usually revolves around addressing management practices that precipitate the development of disease in individual animals. In animals unaccustomed to high-concentrate rations, access to easily digestible feedstuffs should be restricted. Sudden changes in feedstuffs should be avoided and, if changes are made, they should be done gradually, as adaptation of the rumen microbes to new feeds might take several weeks. Bunk space must be adequate to allow all animals to access feed without excessive competition. Similarly, animals fed from a bunk should have feed given to them at consistent intervals. If animals are fed from a self-feeder, it is essential to ensure that adequate roughage is available and feed is increased gradually. In addition, monitoring the level of feed in self-feeders is important to ensure animals do not become excessively hungry and gorge themselves when feed is again available. Feed additives such as ionophore antimicrobials, bicarbonate, and limestone might reduce disease severity and incidence when used appropriately.

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